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ORIGINAL ARTICLE

The associations between anthropometric measurements and left ventricular structure and function: the Echo-SOL Study

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Summary

Objective

The objective of this study is to determine associations between anthropometry and echocardiographic measures of cardiac structure and function in Hispanic/Latinos.

Methods

A total of 1,824 participants from ECHO-SOL were included. We evaluated associations between echocardiographic measures of left ventricular structure and function and anthropometric measures using multivariable-adjusted linear and logistic regression models adjusting for traditional cardiovascular risk factors.

Results

The mean age was 56 ± 0.17 years, 57% were women. The mean body mass index (BMI) was $30 \pm 9.4 \text{ kg m}^{-2}$, waist circumference (WC) was $100 \pm 18 \text{ cm}$, and waist-to-hip ratio (WHR) was 0.93 ± 0.15 . Adjusted analysis showed that 5-unit increment in BMI and 5-cm increase in WC was associated with 3.4 ± 0.6 and $1.05 \pm 0.05 \text{ g m}^{-2.7}$ ($p < 0.05$ for both) higher left ventricular (LV) mass index, respectively. Similarly, 0.1-unit increment in WHR was associated with $2.0 \pm 0.16 \text{ g m}^{-2.7}$ higher LV mass index ($p < 0.01$). WHR was associated with $0.22 \pm 0.08\%$ decrease in ejection fraction ($p < 0.05$). Concomitantly, 5-unit increment in BMI and WC was associated with increased odds of abnormal LV geometry (odds ratio 1.40 and 1.16, $p = 0.03$ and < 0.01 , respectively); 0.1-unit increment in WHR was associated with increased odds of abnormal LV geometry (odds ratio 1.51, $p < 0.01$).

Conclusions

Among Hispanic/Latinos, higher anthropometric measures were associated with adverse cardiac structure and function.

Keywords: anthropometrics, cardiac structure and function, Hispanics.

Introduction

Obesity is an independent risk factor for the development of heart failure (HF) (1–4). The increasing prevalence of HF, in particular, HF with preserved ejection fraction in the setting of the obesity pandemic (5,6), is a significant and burgeoning public health problem (7,8). Hispanic/Latinos are a fast-growing population with a high prevalence of obesity (9). The overall unadjusted prevalence of obesity among Hispanic adults from National Health and Nutrition Examination Survey data

between 2014 and 2015 was 42.7% (10). This estimate makes Hispanic/Latinos the second leading ethnic group with obesity in the United States.

Concomitantly, there is accumulating evidence to support a predisposition for left ventricular (LV) dysfunction and HF among Hispanic/Latinos (11). In this regard, Mehta *et al.* showed that 50.3% of individuals from various Hispanic/Latino backgrounds have echocardiographic evidence of LV diastolic dysfunction (12). Similarly, higher prevalence of LV hypertrophy and abnormal LV remodelling has been observed in different

Hispanic/Latino groups compared with non-Hispanic Whites, even after adjusting for hypertension (13).

Obesity increases the risk of developing diabetes (14), hypertension (15) and dyslipidaemia, all major risk factors for the development of coronary heart disease (16,17). Coronary heart disease is an important cause of HF (18). In addition, both hypertension and diabetes are independent risk factors for the development of HF. Furthermore, obesity may lead to undesirable left ventricular remodeling (19). Left ventricular chamber dilatation is observed due to the chronic elevation of filling pressures and volume overload in obese individuals (20). Left ventricular hypertrophy (LVH), concentric remodeling and concentric LVH are associated with obesity independent of age and arterial pressure (20). In addition to structural abnormalities, obesity has adverse effects on systolic and diastolic functions (21,22).

Preclinical echocardiographic findings of LV dysfunction in populations affected by overweight and obesity can serve as a fundamental step to develop effective HF preventive interventions. Hispanic/Latinos have been identified as a group at risk for HF (11) and have high rates of obesity (23). However, there is a lack of data evaluating the associations between anthropometric measures and left ventricular structure and function in US Hispanic/Latinos. Therefore, we aimed to determine the associations between different anthropometric and body composition variables with echocardiographic measures of LV structure and function in a diverse cohort of US Hispanic/Latino adults from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). The hypothesis for this analysis was that higher levels of adiposity are significantly associated with larger LV mass and volumes but smaller ejection fraction and cardiac output. Also, that fat free mass will be significantly associated with larger LV mass and volumes, as well as ejection fraction and cardiac output.

Methods

Population

The HCHS/SOL is a multicentre community-based, prospective cohort study of 16,415 self-identified Hispanics ages 18–74 recruited from four field centres in Bronx, NY; Chicago, IL; Miami, FL; and San Diego, CA. The HCHS/SOL was designed to examine the risk and protective factors for chronic diseases and associated morbidity and mortality among Hispanic/Latinos in the United States. The HCHS/SOL is composed of individuals from the following heritage: Central American ($n = 1,732$), Cuban ($n = 2,348$), Dominican ($n = 1,473$), Mexican ($n = 6,472$), Puerto-Rican ($n = 2,728$) and South American

($n = 1,072$). Details on the sampling and designs have been published separately (24).

The echocardiographic study of Latinos (ECHO-SOL) is an ancillary study to the HCHS/SOL designed to characterize cardiac structure and function and their determinants. The study population consists of 1,824 participants, approximately 450 individuals from each field centre. Individuals older than 45 years of age who met eligibility criteria for the HCHS/SOL and who had completed all baseline examinations were eligible participants for ECHO-SOL. All echocardiograms for ECHO-SOL were completed after baseline examination but within 36 months of initial collection of clinical and laboratory data. Stratified random sampling was used, drawing participants from the communities representing the four field centres to provide a population sample representative of the HCHS/SOL population and the Hispanic subgroups characteristic of the field centres. Details on the methodology for the ECHO-SOL study have been published (25).

Echocardiographic measurements

ECHO-SOL field imaging centres were established using existing echocardiographic equipment from the HCHS/SOL at each of the four field centres. Phillips Ultrasounds IE-33 (Philips Health Care, Andover, MA, USA) or Sonos 5500/7500 ultrasound imaging platform were used in all field imaging centres. Under a standardized protocol and after completion of in-person training, experienced sonographers in each field centre completed standard echocardiographic examinations including two-dimensional imaging with harmonics spectral Doppler and tissue Doppler. Digital echocardiographic images were electronically transferred from the field centres to the Core Lab at Wake Forest School of Medicine. All ECHO-SOL echocardiograms were read by a cardiologist with experience in echocardiography (Dr Rodriguez). In order to confirm accuracy of key quantitative measures, echocardiogram over-reads were performed. Inter and intra-reader reproducibility was assessed and previously published (25). Intra-class correlations for inter-reader reproducibility ranged from 0.80 to 0.99 with left atrial volume and LV end-diastolic volumes having the highest intra-class correlation values (0.97–0.99).

LV chamber size and wall thickness linear dimensions were used to calculate LV mass (LVM). LVM was indexed (LVMI) to height^{2.7}. Relative wall thickness (RWT) was defined as $2 * (\text{LV Posterior Wall Thickness} / \text{LV internal diameter at diastole})$. The bi-plane method of disc (Simpson's method) was used to obtain LV volumes, and ejection fraction (EF) was calculated from the formula $\text{EF} = \text{EDV} - \text{ESV} / \text{EDV}$; where EDV and ESV are the

volumetric measurements of LV end-diastolic and end-systolic volumes, respectively. LV stroke volume (SV), reported in mL, was assessed utilizing LV outflow tract (LVOT) dimensions, measured in cm^2 and pulse wave spectral Doppler for LV outflow tract Velocity Time Integral. Cardiac output (CO) is the SV multiplied by heart rate. LV diastolic function was assessed with pulse wave mitral inflow velocities (E and A), tissue Doppler mitral annular velocities (e' septal and e' lateral) and left atrial volume (LAV) measured in biplane views index to body surface area. LV diastolic function was categorized as Grade I (mild) to Grade III (severe) according to published ASE (American Society of Echocardiography) and Redfield definitions (26). Left ventricular geometry was categorized as normal, concentric remodelling, concentric hypertrophy and eccentric hypertrophy using LVMI^{2.7} and RWT. The cuff off value for normal LVMI^{2.7} was 45 and for RWT was 0.42 (27).

Anthropometric measurements

The participants wore light clothing and slippers for all measurements and were barefoot when measuring weight and body composition. Standing height was measured with a fixed stadiometer with a vertical backboard and moveable headboard. Weight and body composition was measured with the Tanita Body Composition Analyser, TBF-300A (Tanita Corporation of America, Inc., Arlington Heights, IL, USA). Body mass index (BMI) was calculated averaging two body weight measurements and two standing height measures. The World Health Organization BMI classification was used: normal = $18.5\text{--}25 \text{ kg m}^{-2}$, overweight = $25\text{--}29.99 \text{ kg m}^{-2}$ and obese $\geq 30 \text{ kg m}^{-2}$. Waist circumference (WC) was measured at the level of the upper border of the iliac crest. WC measurement was done using Gulick II 150 and 250 anthropometric tapes (Sammons Preston, Chicago, IL, USA). Hip circumference (HC) was measured at the level of maximum protrusion of the buttocks. Waist-to-hip ratio (WHR) was defined as the WC divided by the HC. The Tanita scale used bioelectrical impedance to measure the participant's total body fat percent, fat mass (FM) and lean body mass to calculate the fat-free mass (FFM).

Clinical covariates

All variables were obtained using methods that have been previously described (24). Briefly, an interviewer-administered questionnaire was used to assess participants' medical history. Participants were fasting 12 h before providing venous blood samples. Laboratory measurements from blood included total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides,

glucose and creatinine. Dyslipidaemia was defined as total cholesterol of 240 mg dL^{-1} or greater, low-density lipoprotein cholesterol 160 mg dL^{-1} or greater or HDL cholesterol less than 40 mg dL^{-1} , or receiving cholesterol-lowering medication. Diabetes was defined as a fasting plasma glucose level of 126 mg dL^{-1} or greater, 2 h post-load glucose 200 mg dL^{-1} or greater, or A1c of 6.5 or greater or taking antihyperglycaemic medication. Normal range plasma creatinine was $0.5\text{--}1.2 \text{ mg dL}^{-1}$ for men and $0.4\text{--}1.0 \text{ mg dL}^{-1}$ for women (28). Three-seated blood pressure measurements were obtained with an automatic sphygmomanometer; the second and third reading were averaged. Hypertension was defined as a systolic blood pressure $\geq 140 \text{ mmHg}$, or diastolic blood pressure $\geq 90 \text{ mmHg}$ or taking blood pressure medication.

Data analysis

Continuous variables were presented as means (SD) and categorical variables as counts (percentage). Continuous variables were compared using simple regression analysis across categories of BMI. Categorical variables were compared using Rao–Scott Chi-square test across categories of BMI. We tested associations between various anthropometric parameter (BMI, WC, HC, WHR, FM and FFM) and the echocardiographic parameter of cardiac structure and function using multivariable linear regression models. Using multivariable logistic regression, we also tested the association between anthropometric measures and diastolic dysfunction and left ventricular geometry. Diastolic dysfunction was defined as normal or abnormal (class I–III), and left ventricular geometry was normal or abnormal (if concentric remodelling, eccentric remodelling or concentric hypertrophy was identified).

The covariates, for both multivariable linear and logistic regression, included age, gender, height (except when BMI was the predictor variable), FFM (except when FFM was the predictor variable), hypertension and taking antihypertensive medications, diabetes, dyslipidaemia, smoking and family history of cardiac disease. Statistical significance was defined as a p value < 0.05 . Survey methods were utilized, and all reported values were weighted to adjust for sampling probability and nonresponse bias to make the inference applicable to the ECHO-SOL target population. All analyses were performed using PROC SURVEY with SAS version 9.3 (SAS Institute Inc, Cary, NC).

Results

The average age was 56 ± 0.17 years, and 57% of participants were women. Mean values for blood pressure (systolic blood pressure $136 \pm 38 \text{ mmHg}$ and diastolic blood

pressure 78 ± 34 mmHg), HDL cholesterol (50 ± 17 mg dL⁻¹) and serum creatinine (0.8 ± 0.13 mg dL⁻¹) were within the normal range. The mean BMI was 30 ± 9.4 kg m⁻², WC = 100 ± 18 cm, fat mass = 29 ± 16 kg, and fat-free mass = 50 ± 13 kg. The mean LVEF was $60 \pm 8.5\%$, LVMI = 42 ± 15 g m^{-2.7}, ESV = 34 ± 17 mL, EDV = 84 ± 37 mL and CO = 5 ± 2.1 L min⁻¹. Please refer to Table 1.

As shown in Table 2, among those with obesity, 67% were women, those with overweight included 51% women compared with 48% women in those with normal BMI (p value < 0.0001). People with obesity had lower rates of smoking compared with individuals with normal BMI (13% vs. 29%, p < 0.0001). The proportion of participants with diabetes, hypertension and dyslipidaemia was higher in individuals with obesity (37%, 56.2% and 44.3%) and overweight (23.1%, 46.3% and 40.9%) compared with those with a normal BMI (17.5%, 41.6% and 31.4%).

Mean echocardiographic measures of LVMI, ESV, EDV, SV and CO were higher among those with obesity (45.2 g m^{-2.7}, 35 mL, 86.8 mL, 73 mL and 4.8 L)

compared with those with normal BMI (34.7 g m^{-2.7}, 32.4 mL, 79.4 mL, 65.7 mL and 4.2 L, and 79.4 mL, p value < 0.05 for all comparisons) The lateral and septal E/E' was higher for those with obesity (9.3 and 12.5 m s⁻¹) compared with people with normal BMI (8.2 and 10.7 m s⁻¹; p value < 0.0001 for both comparisons). The rate of eccentric remodelling and concentric hypertrophy was higher in people with obesity (16.8% and 21.2%) than those with normal BMI (2.7% and 3.8%; p value < 0.0001). Furthermore, the rate of normal LV geometry was higher in those with normal BMI compared with those with obesity (76.9% and 46.4%; p value < 0.0001). Diastolic dysfunction grade 1 and grade 2 were higher among people with obesity (17.4% and 48.6%) and overweight (19.7% and 37.4%) compared with the normal BMI group (12.2% and 30.3%; p value = 0.0001). Similar diastolic dysfunction, the proportion of individuals with normal LV geometry was lower in people with obesity compared with those with normal BMI (46.4% and 76.9%; p value < 0.0001).

In linear regression models adjusted for age, gender, FFM and cardiovascular risk factors, every 5-unit increment in BMI was associated with a 3.4 g m^{-2.7} increase in LVMI (p < 0.05), a 101.5 -mL increase in CO (p < 0.05) and a 0.45 higher E/E' ratio (p < 0.05). Similarly, for every 5-cm increase in WC was associated with a 1.05 g m^{-2.7} increase in LVMI (p < 0.01), a 0.15 -mL increase in ESV (p < 0.02), a 0.40 -mL increase in EDV (p < 0.05), a 43.5 mL min⁻¹ increase in CO (p < 0.01), a 0.35 -mL increase LAV (p < 0.01) and a 0.1 higher E/E' ratio (p < 0.01). Similar findings were noted for HC, FM, and FFM, please refer to Table 3. However, each 0.1-unit increment in WHR was associated with a 2.0 g m^{-2.7} increase in LVMI (p < 0.01), and a 0.22 decrease in EF (p < 0.01), as well as a 0.66 -mL decrease in ESV (p < 0.01) and 1.71 -mL decrease in EDV (p < 0.01).

To summarize, BMI, WC, HC, FM and FFR were all associated with higher LVMI, CO, LAV and E/E' independent of age, sex, hypertension and antihypertensive medication use, diabetes, dyslipidaemia, smoking and family history of coronary heart disease. In the other hand, WHR was associated with higher LVMI but lower ESV, EDV and EF.

In multivariable logistic regression models (Table 4), every 5 kg m⁻² increase in BMI was associated with higher odds of abnormal left ventricular geometry (odds ratio, OR 1.40, 95% confidence interval, CI [1.3, 1.9]) and diastolic dysfunction (OR 1.48, 95% CI [1.2, 1.8], p < 0.01). Similarly, each 5-cm increase in WC was associated with increased odds of abnormal left ventricular geometry (OR 1.17, 95% CI [1.09, 1.26]) and increased odds of diastolic dysfunction (OR 1.1, 95% CI [1.01, 1.2]). Each 0.1-unit increase in WHR was associated with higher odds of

Table 1 Baseline characteristics

	Mean (SE)
Age (years)	56 ± 0.17
SBP (mmHg)	136 ± 38
DBP (mmHg)	78 ± 34
Heart rate (bpm)	66 ± 24
HDL cholesterol (mg dL ⁻¹)	50 ± 17
Serum Cr (mg dL ⁻¹)	0.8 ± 0.13
BMI (kg m ⁻²)	30 ± 9.4
Waist circumference (cm)	100 ± 18
Hip circumference (cm)	107 ± 21
Waist-to-hip ratio	0.93 ± 0.15
Fat mass (kg)	29 ± 16
Fat free mass (kg)	50 ± 13
Height (cm)	163 ± 17
	<i>N</i> (%)
Women (%)	882 (57)
Diabetes (%)	523 (28)
Current smokers (%)	210 (18)
Echocardiographic parameters	Mean (SE)
Left ventricular mass index (g m ^{-2.7})	42 ± 15
End systolic volume (mL)	34 ± 17
End diastolic volume (mL)	84 ± 37
Ejection fraction (%)	60 ± 8.5
Fractional shortening (%)	30 ± 12
Cardiac output (L min ⁻¹)	5 ± 2.1
Lateral peak systolic velocity (cm s ⁻¹)	8 ± 4.3

Data are presented as mean \pm SD or *N* (%) using weighted row percentages.

BMI, body mass index; DBP, diastolic blood pressure; HDL, high-density lipoprotein; SBP, systolic blood pressure; SE, standard error.

Table 2 Unadjusted associations between baseline characteristics and BMI

Clinical and echo characteristic	BMI								Overall <i>p</i> value
	<25 (<i>N</i> = 225)		25–29 (<i>N</i> = 529)			>30 (<i>N</i> = 598)			
	Mean	SE	Mean	SE	<i>p</i> value	Mean	SE	<i>p</i> value	
Age (years)	56	0.73	57	0.46	0.68	56	0.63	0.82	<0.0001
Lean body mass (kg)	46	0.6	49	0.48	0.0007	52	0.53	<0.0001	<0.0001
Fat mass (kg)	16	0.29	24	0.19	<0.0001	38	0.5	<0.0001	<0.0001
WC (cm)	85.5	0.61	95	0.33	<0.0001	109.1	0.69	<0.0001	<0.0001
SBP (mmHg)	133.3	0.43	135.4	0.99	0.24	137.9	1.07	0.01	<0.0001
DBP (mmHg)	74.8	0.76	77.7	0.68	<0.01	79.2	0.49	<0.0001	<0.0001
HDL (mg dL ^{−1})	55.5	1.27	54.8	0.59	<0.01	48.3	0.53	<0.0001	<0.0001
Creatinine (mg dL ^{−1})	0.94	0.06	0.86	0.015	0.21	0.8	0.01	0.03	<0.0001
LVMl (g m ^{−2.7})	34.7	0.57	40.1	0.53	<0.0001	45.2	0.55	<0.0001	<0.0001
LVPWd (cm)	0.81	0.01	0.89	0.01	<0.0001	0.91	0.01	<0.0001	<0.0001
IVSd (cm)	1	0.01	1	0.01	<0.0001	1.11	0.01	<0.0001	<0.0001
RWT	0.37	0.01	0.37	0.01	<0.0001	0.4	0.01	<0.0001	<0.0001
ESV, mean	32.4	0.88	32.9	0.59	0.59	35	0.48	0.01	<0.0001
EDV, mean	79.4	1.89	81.5	1.07	0.31	86.8	1.02	<0.01	<0.001
EF, mean	59.4	0.4	59.9	0.29	0.2	59.8	0.25	0.32	<0.0001
SV (LVOT) (mL)	65.7	1.1	69.1	0.97	0.02	73	0.95	<0.0001	<0.0001
CO (L)	4.2	0.079	4.5	0.071	<0.01	4.8	0.06	<0.0001	<0.0001
E/E' lateral ratio (mean)	8.2	0.18	8.7	0.27	0.04	9.3	0.29	<0.0001	<0.0001
E/E' septal ratio (mean)	10.7	0.25	11.1	0.19	0.29	12.5	0.2	<0.0001	<0.0001
LA volume	39.6	1.15	42.4	0.73	0.04	43.5	0.6	0.001	<0.001
	Percent		Percent			Percent			<i>p</i> value
Women	48		51			67			<0.0001
Current smoker	29		18			13			<0.0001
Current alcohol use	47		49			38			0.014
Diabetes	17.6		23.1			37			<0.0001
Hypertension	41.5		46.3			56.2			0.0024
Dyslipidaemia	32.2		40.9			44.3			0.05
Left ventricular geometry									<0.0001
Normal (%)	76.9		56.8			46.4			
CR (%)	16.6		23.3			15.6			
ER (%)	2.7		7.7			16.8			
CH (%)	3.8		12.2			21.2			
LV diastolic function									<0.0001
Normal	55.4		41.1			31.8			
Grade 1 (%)	12.2		19.7			17.4			
Grade 2 (%)	30.3		37.4			48.6			
Grade 3 (%)	2.1		1.8			2.2			

Data are presented as mean or *N* (%) using weighted row percentages; *N*'s presented are unweighted counts of total participants in the HCHS-SOL with respective characteristic.

BMI, body mass index; CO, cardiac output; CR, concentric hypertrophy; DBP, diastolic blood pressure; EDV, end-diastolic volume; EF, ejection fraction; ER, eccentric hypertrophy; ESV, end-systolic volume; HDL, high-density lipoprotein; IVSd, interventricular septum thickness; LA, left atrial; LV, left ventricular; LVMl, left ventricular mass index; LVOT, left ventricular outflow tract; LVPWd, left ventricular posterior wall thickness; RWT, relative wall thickness; SBP, systolic blood pressure; SE, standard error; SV, stroke volume; WC, waist circumference.

abnormal left ventricular geometry (OR 1.51, 95% CI [1.12, 2.03]), but WHR was not significantly associated with diastolic dysfunction (OR 1.18, 95% CI [0.93, 1.48]). Every 1-kg increase in FM and FFM was associated higher odds of abnormal left ventricular geometry (OR 1.03, 95% CI [1.0, 1.05] and OR 1.02, 95% CI [0.98, 1.05], respectively) and higher odds of diastolic

dysfunction (OR 1.02, 95% CI [1.0, 1.04] and OR 1.06, 95% CI [1.02, 1.10]).

In summary, BMI, WC, WHR and FM were associated with statistically significant higher odds of abnormal left ventricular geometry. Similarly, BMI, WC, FM and FFM were associated with higher odds of diastolic dysfunction.

Table 3 Multivariable linear regression models of the associations between anthropometric measures and echocardiographic parameters

Predictor	LVMI		ESV		EDV		EF		CO		LAV		E/E'	
	B (SE)	R ²	B (SE)	R ²	B (SE)	R ²	B (SE)	R ²	B (SE)	R ²	B (SE)	R ²	B (SE)	R ²
BMI	3.4 (0.60)*	0.21	0.5 (0.45)	0.3	1.75 (1)	0.34	0.1 (0.20)	0.06	101.5 (49.5)*	0.19	0.3 (0.6)	0.12	0.45 (0.12)*	0.21
WC	1.05 (0.05)*	0.22	0.15 (0.05)*	0.3	0.40 (0.1)*	0.34	-0.05 (0.04)	0.07	43.5 (12.5)*	0.19	0.35 (0.1)*	0.12	0.10 (0.01)*	0.21
HC	0.55 (0.04)*	0.2	0.30 (0.05)*	0.3	0.85 (0.1)*	0.35	-0.01 (0.03)	0.07	38 (11.5)*	0.19	0.25 (0.05)*	0.12	0.10 (0.01)*	0.21
WHR	2.00 (0.16)*	0.2	-0.66 (0.13)*	0.3	-1.71 (0.29)*	0.35	-0.22 (0.08)*	0.07	29.3 (18.5)*	0.19	0.32 (0.23)	0.12	0.05 (0.05)	0.20
FM	0.19 (0.01)*	0.21	0.08 (0.01)*	0.3	0.26 (0.03)*	0.35	-0.001 (0.01)	0.07	12.5 (3.7)*	0.19	0.1 (0.02)*	0.12	0.02 (0.00)*	0.21
FFM	0.41 (0.06)*	0.15	0.52 (0.07)*	0.3	1.2 (0.16)*	0.34	-0.01 (0.03)	0.06	68.0 (8.3)*	0.19	0.57 (0.08)*	0.12	0.04 (0.01)*	0.19

Model adjusted for age, gender, height (except for BMI models where height has been omitted), FFM (except for FFM models), HTN, DM, dyslipidaemia, smoking and family history of coronary heart disease.

**p* value <0.05.

BMI, body mass index; CO, cardiac output; DM, diabetes mellitus; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; FFM, fat-free mass; FM, fat mass; HC, hip circumference; HTN, hypertension; LAV, left atrial volume; LVMI, left ventricular mass index; SE, standard error; WC, waist circumference; WHR, waist-to-hip ratio.

Table 4 Multivariable logistic regression models of the associations between anthropometric measures and echocardiographic parameters

	LV geometry			Diastolic dysfunction		
	Odds ratio	95% CI	<i>p</i> value	Odds ratio	95% CI	<i>p</i> value
BMI	1.40	[1.30, 1.90]	0.03	1.48	[1.20, 1.80]	<0.01
WC	1.17	[1.09, 1.26]	<0.01	1.10	[1.01, 1.20]	0.04
HC	1.05	[0.94, 1.17]	0.41	1.07	[0.98, 1.17]	0.13
WHR	1.51	[1.12, 2.03]	<0.01	1.18	[0.93, 1.48]	0.22
FM	1.03	[1.00, 1.05]	0.02	1.02	[1.0, 1.04]	0.05
FFM	1.02	[0.98, 1.05]	0.42	1.06	[1.02, 1.10]	0.03

Model 1: Age, gender, height (except for BMI models where height has been omitted), FFM (except FFM which is adjusted for FM), HTN, DM, dyslipidaemia, smoking and family history of coronary heart disease.

Left ventricular geometry is categorized as normal versus abnormal. The abnormal category includes left ventricular eccentric remodelling, eccentric hypertrophy and concentric hypertrophy.

BMI, body mass index; CI, confidence interval; FFM, fat-free mass; FM, fat mass; HC, hip circumference; WC, waist circumference; WHR, waist-to-hip ratio.

Discussion

In this cross-sectional analysis of the association between anthropometrics, body composition measures and echocardiographic measures of left ventricular structure and function in Hispanic/Latinos, we found that higher anthropometrics and body composition measures were associated with undesirable cardiac structure and function. Specifically, BMI, WC, HC, FM and FFM were associated with higher LVMI, CO and E/E', while WHR was significantly associated with higher LVMI and lower

ESV, EDV and lower EF. Similarly, BMI, WC and FM were associated with abnormal left ventricular geometry (eccentric remodelling, eccentric hypertrophy and concentric hypertrophy) and diastolic dysfunction (grades I–III). These associations were independent of age, sex, diabetes, hypertension, dyslipidaemia, smoking and personal and family history of cardiovascular disease.

Hispanic/Latinos are a fast-growing minority in the USA with high prevalence of obesity and other cardiovascular risk factors (9) that place them at increased risk for the development of HF (11). Our study shows that higher anthropometric measures are associated with higher LVMI and CO but lower EF except for BMI for which EF is higher. Also, higher anthropometrics are associated with abnormal left ventricular geometry (concentric remodelling, concentric hypertrophy and eccentric hypertrophy) after adjusting for traditional cardiovascular risk factors including hypertension, dyslipidaemia, smoking and personal and family history of cardiovascular disease. Abnormal LV geometry is commonly observed in patients with normal LV systolic function. Unfortunately, abnormal LV geometry with or without LVH is associated with adverse outcomes. Older and obese patients are more commonly found to have abnormal LV geometry patterns (29).

Obesity can influence the development of left ventricular hypertrophy, and therefore, abnormalities in left ventricular geometry through increase haemodynamic load (3) and inflammation THAT promote adverse cardiac remodeling (30). Hypertension is strongly correlated with obesity (15), and hypertension is known to be associated with eccentric left ventricular hypertrophy. Although it is difficult to separate the effect of hypertension from that of obesity, we included hypertension in our linear and logistic regression models to account for this known

association. Similarly, obesity and diabetes are strongly associated (31), and both are pro-inflammatory conditions that could lead to adverse cardiac remodelling (high LVMI) (30). Diabetes was therefore included in our models as a confounder for the association between obesity and cardiac structure. Our final results demonstrate an independent association between obesity and higher LVMI and abnormal left ventricular geometry after adjusting for major confounders such as hypertension and diabetes.

Higher BMI, WC, HC, FM and FFM were associated with higher E/E' and left ventricular diastolic dysfunction independent of age, sex and common cardiovascular risk factors. Left ventricular diastolic dysfunction is a finding related to abnormal cardiac filling pressure during the relaxation phase of the cardiac cycle and is associated with an increased risk of future HF risk (32). Similarly, E/E' is a widely used indicator of left ventricular filling pressures and predictor of HF risk (33,34). While previous studies have documented the association between obesity and abnormal cardiac structure and function (21,35–38), there is lack of data describing these associations in US Hispanic/Latinos. Among the existing studies, the sample might not be representative or the US Hispanic/Latino population (39). Our study provides data to fill in this gap and serves as the groundwork to inform prospective and intervention studies aimed to determine the use of echocardiography as a risk stratification tool for HF in US Hispanic/Latinos affected by overweight and obesity.

Our study limitations include recruitment of specific Hispanic/Latino background groups by study site because of differential geographic distribution among Hispanic/Latino groups residing in the United States. The ECHO-SOL echocardiograms were **not** contemporaneous with data from the HCHS/SOL. However, the time intervals between ECHO-SOL assessment and HCHS/SOL measurements were reduced to the best possible, and the most updated HCHS/SOL data on key covariates (hypertension, diabetes mellitus and self-reported HF) were used. The cross-sectional nature of the analysis limits causal inference and is prone to potential reverse causality. Furthermore, our study lacks clinical outcomes.

The strengths of this study include the use of a large and diverse US Hispanic/Latino population sample with standardized comprehensive anthropometric and echocardiographic measures of cardiac structure and function. We provide information on the association between various measures of adiposity rather than limiting to commonly used BMI and WHR. The inclusion of WC, HC, FM and FFM allows for assessment of different body fat distribution patterns important to the population studied.

Conclusion

Understanding anthropometrics and body composition measures and their relationship to echocardiographic markers of left ventricular dysfunction aid in identifying populations at risk for HF. The findings from this cross-sectional analysis contribute the first comprehensive anthropometric and body composition analysis reporting the associations with echocardiographic measures of LV structure and function in a large cohort of US Hispanic/Latinos.

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Conflict of Interest Statement

The authors declare no conflict of interest.

Author Contributions

Dr Ponce interpreted the data and drafted the manuscript. Katrina Swett completed the analyses for this study. Dr Allison conceived the study, develop the manuscript proposal and interpreted the data. Dr Cai, Desai, Hurwitz, Ni, Schneiderman, Shah, Spevack, Talavera and Rodriguez contributed to the methodological aspects of this study. All authors were involved in the manuscript writing and approved the final version submitted for publication.

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